

AMENDMENTS TO THE CLAIMS

Please enter the following amendments to the claims without prejudice, without admission, without surrender of subject matter, and without any intention of creating any estoppel as to equivalents as follows:

1. (Currently amended) A transgenic mouse model showing hypomyelinosis of the thalamus of oligodendrocyte developmental disorders wherein the transgenic mouse comprises a homozygous disruption in chromosomal DAP12 (DNAX Activation Protein 12) gene function, and wherein the transgenic mouse shows hypomyelinosis of the thalamus homozygous disruption includes the promoter region and exons 1, 2, and 3.

2. (Canceled)

3. (Currently amended) The transgenic mouse model of claim 1, wherein the homozygous disruption in DAP12 can be pheonotypically exhibited as oligodendrocyte developmental disorder is a myelinogenesis developmental disorder or a neuropsychiatric disorder.

4. (Currently amended) The transgenic mouse model of claim 3, wherein the neuropsychiatric disorder is selected from the group consisting of Nasu-Hakola disease, dementia, schizophrenia, schizotypal personality disorders, obsessive-compulsive disorders, Huntington's disease or Tourette's syndrome.

5. (Currently amended) The transgenic mouse model of claim 3, wherein the neuropsychiatric disorder is Nasu-Hakola disease or dementia.

6-18. (Canceled)

19. (Previously presented) The transgenic mouse model of claim 1, wherein the expression of myelin basic protein in the brain is weak in regions where DAP12 is strongly expressed in wild-type mice.

20. (Previously presented) The transgenic mouse model of claim 1, wherein the transgenic mouse exhibits an impairment in sensorimotor gating as compared to wild-type mice.